THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM OPIOIDS

Using the Recovery Kernel Framework

Heroin · Fentanyl · Oxycodone · Hydrocodone · Morphine · Codeine · Tramadol · Kratom · Prescription Opioids

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

CARRY NALOXONE. IF YOU OR SOMEONE YOU KNOW USES OPIOIDS, NALOXONE SAVES LIVES.

Available without prescription at most US pharmacies. Get it before you need it.

A Note Before You Begin

This guide is different from any other in this series in one essential way: the opioid epidemic is an active mass casualty event. While you are reading this sentence, someone is dying of an opioid overdose. The rate in the United States alone exceeds 100,000 deaths per year. Fentanyl has contaminated the drug supply so thoroughly that no street opioid can be assumed safe regardless of what it is sold as.

This guide therefore opens not with biology but with the most urgent harm reduction information that exists — because the framework in these pages is of no use to someone who does not survive long enough to use it.

After the immediate safety section, this guide addresses what the Recovery Kernel is built to address: the specific biology that made opioids feel necessary, and the systematic reconstruction of the systems they damaged. The compendium's framework applies powerfully to opioid recovery. But nothing in it matters if you are not alive.

A note on medication-assisted treatment (MAT): this guide takes the position the Recovery Kernel Compendium takes, stated plainly — buprenorphine and methadone are life-saving medications. Refusing them because 'it is still drugs' has killed people who could have been saved. The person maintained on MAT who is functioning, connected, and building a life is in genuine recovery, regardless of what

anyone else says about it. This guide supports MAT as a foundation, not a compromise.

Section 0 — Immediate Safety: Read This First

⚠ IF YOU ARE IN AN OVERDOSE EMERGENCY: CALL 911 NOW. GIVE NALOXONE IF AVAILABLE. DO NOT LEAVE THE PERSON ALONE.

Naloxone (Narcan) — The Life-Saving Reversal

THE CENTURION — The Single Most Important Harm Reduction Tool in the Opioid Crisis

Naloxone is an opioid receptor antagonist that reverses opioid overdose within minutes. It is safe, effective, available without a prescription at most US pharmacies, and has no effect on someone who has not taken opioids. There is no reason not to have it.

Naloxone form	How to use	Important notes
Narcan nasal spray (most common)	Lay person on back. Give one spray in one nostril. If no response in 2–3 minutes, give second dose in other nostril. Repeat every 2–3 minutes until emergency services arrive.	Effects last 30–90 minutes — shorter than most opioids. Person may re-enter overdose when naloxone wears off. Stay with them until EMS arrives. Call 911 first, then administer.
Injectable naloxone (vial + syringe)	Draw 1ml (0.4mg) into syringe. Inject into muscle (outer thigh, upper arm). Repeat every 2–3 minutes if no response.	Faster onset than nasal spray. Requires syringe. Less accessible but effective.
Kloxxado / Rextovy (higher dose nasal sprays)	Same as Narcan nasal spray. Higher dose formulations designed for fentanyl overdose where standard doses may be insufficient.	With fentanyl, multiple doses of standard naloxone may be needed. High-dose formulations reduce this risk. Ask your pharmacist specifically for fentanyl-appropriate naloxone.

Recognizing an Opioid Overdose

- Unresponsive or cannot be woken — will not respond to voice or sternal rub
- Slow, shallow, or stopped breathing — fewer than one breath every 5 seconds
- Choking, gurgling, or snoring sounds — airway obstruction from muscle relaxation
- Blue or gray lips, fingertips, or fingernails (cyanosis) — oxygen deprivation

- Pinpoint pupils — very small, contracted pupils even in dim light
- Limp body

IF ANY OF THESE ARE PRESENT: Call 911 immediately. Give naloxone. Perform rescue breathing if trained. Place in recovery position (on side) if breathing but unconscious. Do not leave alone.

Fentanyl Test Strips — The Supply Safety Reality

THE CENTURION — Every Street Drug Must Be Assumed to Contain Fentanyl

Fentanyl is 50–100 times more potent than morphine and is now present in heroin, counterfeit prescription pills, cocaine, methamphetamine, and even some cannabis products. A lethal dose of fentanyl is approximately 2 milligrams — invisible to the naked eye. There is no safe assumption about street drugs in 2026.

- Fentanyl test strips: available at many harm reduction organizations, pharmacies, and online. Dissolve a small amount of the substance in water and dip the strip. A positive result means fentanyl is present. A negative result reduces but does not eliminate risk.
- Never use alone: if you are using, have someone present who can administer naloxone and call 911. The 'Never Use Alone' hotline (1-800-484-3731) provides a live person who will call 911 to your location if you stop responding.
- Start low, go slow: if using after a period of abstinence — including after any time in treatment, jail, or hospital — your tolerance is gone. A dose that was normal before can be fatal now. This is the highest-risk period for opioid overdose death.
- Good Samaritan laws: most US states have laws protecting people who call 911 for an overdose from prosecution. Know your state's law. Fear of legal consequences is a primary barrier to calling for help.

Section 1 — What Opioids Did to Your Brain and Body

THE CENTURION — The System That Was Muted — and the Pain That Was Underneath It

The Recovery Kernel Compendium describes opioids with precise language: total sensory muting. Physical agony and existential despair. This is not moralizing. It is mechanistic description of what the opioid receptor system does and what happens when a drug activates it at pharmaceutical scale.

Your brain contains mu, kappa, and delta opioid receptors distributed throughout systems that regulate pain, emotional bonding, stress response, reward, and the basic sense of being at home in your own body. Endorphins — your endogenous opioids — are released during physical exertion, social bonding, laughter, touch, and safety. They are the biological substrate of belonging, comfort, and the feeling that life is bearable.

Opioids activate these receptors with an intensity that endorphins never reach. The pain stops — not just physical pain but the existential pain of being alive in a difficult body, in a difficult world, with a history that may have been genuinely brutal. This is the function that every person reading this guide already knows. The opioid was not solving a fake problem. It was solving a real one. The question the Recovery Kernel asks is: what was the real problem, and can it be addressed another way?

The Three-System Disruption

System	What opioids did	What recovery looks like
Endogenous opioid system (endorphins, enkephalins)	Massively overstimulated mu-opioid receptors. The brain responded by reducing its own endorphin production and downregulating receptor density. The system that generates natural comfort, pain tolerance, and social bonding goes offline.	The most prominent feature of opioid recovery: everything hurts more than it should. Physical pain is amplified. Emotional pain is uncontained. Social connection feels hollow. This is not depression as a character state — it is endorphin system withdrawal. It recovers, but slowly. 6–18 months for full receptor upregulation. The supplement and behavioral protocol must build bridges across this gap.
Dopamine system	Opioids produce indirect dopamine release in the nucleus accumbens. Chronic use downregulates D2	Anhedonia and motivational deficit in opioid recovery reflect both endorphin and dopamine system depletion. The world feels not just painful but colorless and purposeless. This is bilateral system

	receptors. The motivation and pleasure system is secondarily impaired alongside the primary endorphin system damage.	damage. Both systems must be addressed in the reconstruction protocol.
HPA axis (stress response system)	Opioids suppress the HPA axis — cortisol, adrenaline, and the entire stress response is blunted. The body and mind become unable to tolerate normal stress levels.	In withdrawal, the HPA axis rebounds into overdrive — an extreme stress response that had been chemically suppressed for months or years. The profound anxiety, muscle aches, temperature dysregulation, and restlessness of opioid withdrawal are the HPA axis uncaged. Cortisol management and adrenal support become central recovery tools.

The Opioid Class — A Full Spectrum

THE CENTURION — Same Receptor, Different Intensities, Different Recovery Timelines

Substance	Potency relative to morphine	Half-life / withdrawal onset	Recovery-specific notes
Fentanyl / carfentanil (street)	50–10,000x morphine	Short — withdrawal begins 8–24 hrs	THE MOST DANGEROUS. Extreme potency means receptor saturation was extreme — recovery from fentanyl produces more severe withdrawal and longer PAWS than almost any other opioid. The fentanyl supply is also adulterated — carfentanil (10,000x morphine) has been detected. MAT is not optional for most fentanyl-dependent people — it is survival.
Heroin (diacetylmorphine)	2–3x morphine	Short — withdrawal begins 6–12 hrs	Street heroin is now almost universally fentanyl-adulterated or entirely fentanyl. The heroin of 2026 is not the heroin of previous decades. Treat all street heroin as fentanyl for harm reduction and recovery planning purposes.
Oxycodone (OxyContin, Percocet)	1.5x morphine	Medium — withdrawal begins 8–24 hrs	The prescription opioid epidemic's primary driver. Many people in opioid recovery began with legitimate prescriptions. This origin does not change the biology — receptor downregulation is identical to illicit use. The shame architecture may be different: 'I was just taking my

			medication' requires its own identity work.
Hydrocodone (Vicodin, Norco)	Approximately equal to morphine	Medium — withdrawal begins 6–12 hrs	The most prescribed opioid in US history. Combination formulations (with acetaminophen) added liver toxicity risk alongside opioid dependence. NAC and liver support are relevant here.
Morphine / oxymorphone	Reference standard / 3x	Medium — withdrawal 8–24 hrs	Often hospital-origin opioid dependence. Extended-release formulations (MS Contin, Opana ER) produce particularly severe and prolonged withdrawal. Taper under medical supervision before discontinuation.
Methadone (when used non-therapeutically)	Variable — 3–5x morphine equivalent	Very long — withdrawal begins 24–36 hrs; peaks days 3–8; may last weeks	Methadone's extreme half-life (24–36 hours) means withdrawal is prolonged and can peak days after the last dose. When used therapeutically as MAT, this same half-life is what makes it effective — one daily dose provides stable coverage. Never abruptly stop methadone outside of medical supervision.
Tramadol	Approximately 0.1x morphine (but also serotonin/norepinephrine effects)	Medium — withdrawal begins 12–24 hrs	Often underestimated because it is considered 'mild.' Tramadol withdrawal has both opioid AND serotonin/norepinephrine components — producing a more complex and sometimes more distressing withdrawal than pure opioids. Seizure risk exists, particularly with abrupt cessation after high-dose use. CYP2D6 metabolizes tramadol to its active form — poor metabolizers get little opioid effect; ultra-rapid metabolizers may experience toxicity.
Kratom (mitragynine)	Variable — mitragynine is partial mu-agonist	Medium-long — withdrawal begins 12–24 hrs	Kratom is widely marketed as a 'natural' opioid recovery aid and 'safe' alternative to opioids. It is neither. Kratom produces physical dependence with a withdrawal syndrome similar to traditional opioids, plus additional stimulant withdrawal from its adrenergic effects. Long-term heavy kratom users may require MAT for detox. Do not use kratom as a self-taper from other opioids without medical guidance.
Codeine	0.1x morphine	Medium — withdrawal begins 6–12 hrs	CYP2D6 converts codeine to morphine. Ultra-rapid CYP2D6 metabolizers convert dramatically more codeine to morphine — producing toxicity, respiratory depression, and death at doses that are

			safe for others. This is documented, fatal, and genetic. If codeine ever made you feel far more impaired than expected, you may be an ultra-rapid CYP2D6 metabolizer — this is clinically important information.

What Opioids Were Managing — The Compendium's Diagnostic Question

THE CENTURION — The Substance Is the Symptom Report

The Recovery Kernel Compendium makes a precise clinical observation: opioids provide total sensory muting for physical agony and existential despair. This tells you something important about the person who found opioids necessary — not as a moral statement but as a diagnostic one.

The underlying conditions most commonly driving opioid use disorder include:

- Chronic or undertreated physical pain: the opioid began as pain management — legitimate or illegitimate — and the physics of tolerance did the rest. The pain is still there. Recovery requires addressing it directly, not simply removing the medication.
- Trauma stored in the body: complex PTSD, childhood trauma, and adverse childhood experiences (ACEs) produce a specific kind of chronic physical and emotional pain that opioids are uniquely effective at silencing. The trauma does not disappear in recovery — it becomes louder. Protocol-specific trauma work (referenced in Section 7) is not optional for this profile.
- Emotional pain and grief: the specific warmth and social belonging that mu-opioid activation produces means that people who are lonely, grieving, or disconnected may find opioids uniquely compelling. The endorphin deficit that opioids were addressing was real. Building endorphin sources through genuine connection becomes a central recovery target.
- Undertreated psychiatric conditions: depression, anxiety, PTSD, ADHD — all have documented relationships with opioid use disorder as self-medication. Addressing these conditions is part of the biology of recovery, not a separate track.

Knowing which condition you were managing does not excuse the consequences of the addiction. It informs the recovery strategy. An opioid user managing chronic pain needs a pain management protocol. An opioid user managing trauma needs trauma-specific treatment. Generic recovery without this specificity produces the predictable result: the drug is removed but the condition it was managing remains untreated, and the pull back to the substance stays biological rather than merely habitual.

Section 2 — Your Genomic Blueprint

Opioid use disorder has specific genetic architecture that determines both why the substance felt necessary and which recovery strategies will be most effective. The genomic panel for opioid recovery overlaps with the broader Recovery Kernel framework but has specific variants that are uniquely high-stakes in this context.

The DNA Testing Panel for Opioid Recovery

Gene	What it controls	Key variant	Opioid recovery relevance
OPRM1	Mu-opioid receptor structure and sensitivity	A118G (rs1799971)	THE most important gene in opioid recovery. A118G (Asn40Asp) alters the receptor structure — G/G homozygous carriers have significantly reduced opioid receptor binding affinity, meaning they got less pain relief from standard doses (driving dose escalation) AND show dramatically superior naltrexone response. If you carry A118G, present this to your addiction medicine physician — it directly guides treatment selection. Also strongly predicts endorphin-mediated social bonding reward, relevant to natural addiction porting strategy.
CYP2D6	Metabolizes codeine to morphine, tramadol to active form, and many opioid medications	Poor, intermediate, extensive, or ultra-rapid metabolizer	CRITICAL FOR SAFETY AND TREATMENT. Poor CYP2D6 metabolizers get little to no effect from codeine or tramadol (drives dose escalation seeking effect that cannot be achieved). Ultra-rapid metabolizers convert codeine to morphine dangerously fast — associated with death from standard codeine doses. This also affects buprenorphine metabolism and many psychiatric medications used in co-occurring condition treatment. Get tested before MAT dosing optimization.
CYP3A4	Primary metabolizer of fentanyl, oxycodone,	Multiple variants affecting activity	CYP3A4 metabolizes the majority of opioids and both primary MAT medications (buprenorphine and methadone). Inhibited CYP3A4 —

	methadone, buprenorphine		from genetic variants or drug interactions — can cause dangerous accumulation of these medications. CYP3A4 inducers (rifampin, certain anticonvulsants) can precipitate withdrawal from MAT by accelerating metabolism. Inform all prescribers of every medication.
COMT	Dopamine, norepinephrine, and epinephrine clearance	Val158Met (rs4680)	COMT Val158Met affects pain sensitivity — Met/Met (Sage, slow COMT) carriers have higher pain sensitivity and higher endogenous opioid tone at baseline, but also show greater vulnerability to opioid use disorder when exposed to opioids for pain management. Val/Val (Warrior) carriers may underrespond to opioid analgesia at standard doses due to faster dopamine/norepinephrine clearance, driving dose escalation.
MTHFR	Methylation — neurotransmitter synthesis foundation	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants are significantly overrepresented in substance use disorder broadly and opioid use disorder specifically. The chronic serotonin and dopamine deficit from impaired methylation creates a specific kind of low-grade suffering that opioids are highly effective at silencing. 5-MTHF supplementation often produces dramatic early recovery improvement for this profile.
DRD2 / ANKK1	Dopamine D2 receptor density	Taq1A (rs1800497)	A1 allele (reduced D2 density, Reward Deficiency Syndrome) is associated with opioid use disorder through multiple pathways. The baseline 'not enough' experience — the anhedonia and motivational deficit of low D2 density — is powerfully addressed by opioids' secondary dopamine effects. Recovery must build robust reward architecture or the RDS pull will be relentless.
BDNF	Brain-derived neurotrophic factor — neuroplasticity and receptor repair	Val66Met (rs6265)	Met carriers have reduced BDNF secretion. Opioid use further suppresses BDNF. The combination impairs the neuroplasticity required for recovery — new circuit formation, receptor upregulation, and emotional learning all require BDNF. Exercise is the primary BDNF driver and becomes even more essential for Met

			carriers.
ABCB1 (P-glycoprotein)	Blood-brain barrier drug transport — controls how much opioid enters the brain	rs1045642 (C3435T)	ABCB1 variants affect how efficiently opioids cross the blood-brain barrier. Some variants allow greater opioid CNS penetration — producing stronger effects from the same dose, potentially accelerating dependence development. Also affects buprenorphine and methadone CNS concentration — relevant for MAT optimization.

Phenotyping Without Test Results

If genetic testing is not yet available, the compendium's phenotyping tools give working hypotheses:

- Did opioids give you pain relief that no other medication could provide, even at doses others found sufficient? → Possible OPRM1 G/G variant or ABCB1 variant affecting CNS penetration. CYP2D6 testing is important.
- Did opioids make you feel normal, connected, and at ease in a way nothing else achieved? → Strong OPRM1 involvement and likely endorphin system deficit. Social bonding work is central to your recovery architecture.
- Did prescription opioids begin with legitimate pain management? → COMT pain sensitivity variants may be relevant. Undertreated chronic pain requires its own treatment plan alongside opioid recovery.
- Is depression, anhedonia, and a baseline feeling of emptiness your persistent experience? → DRD2 deficit + MTHFR involvement likely. 5-MTHF and dopamine support are high priority.
- Did antidepressants or pain medications respond unpredictably — either not working or working far too intensely? → CYP2D6 or CYP2C19 variant. Inform all prescribers.

Section 3 — Detox, Withdrawal, and Medication-Assisted Treatment

⚠ OPIOID WITHDRAWAL IS NOT FATAL IN MOST CASES — BUT THE RELAPSE RISK DURING WITHDRAWAL IS EXTREME. THE PRIMARY CAUSE OF DEATH IN OPIOID WITHDRAWAL IS NOT THE WITHDRAWAL ITSELF. IT IS RELAPSE FOLLOWED BY OVERDOSE WITH ZERO TOLERANCE.

This distinction matters profoundly. The person suffering through opioid withdrawal is not in immediate danger from the withdrawal itself in most cases. They are in extreme danger from the combination of intolerable suffering and zero tolerance — the person who returns to their prior dose after days of abstinence encounters that dose with the receptor sensitivity of someone who has never used, and dies.

Medical supervision of opioid detox exists not primarily to prevent death from the withdrawal syndrome — though it can address that — but to reduce the suffering to a level where the person does not return to use, and to provide the bridge of MAT that makes sustained recovery possible.

3.1 — Medication-Assisted Treatment: The Foundation, Not the Compromise

THE CENTURION — The Compendium's Position, Stated Without Apology

The Recovery Kernel Compendium states this directly: MAT for opioid addiction consists of life-saving interventions. Refusing them because 'it is still drugs' has killed people who could have been saved.

The evidence is unambiguous. Buprenorphine and methadone reduce opioid overdose deaths by 50–70% in the populations that use them. Nothing else comes close. No combination of supplements, behavioral interventions, willpower, or spiritual practice produces outcomes remotely comparable to MAT for opioid use disorder in the fentanyl era.

This does not mean MAT is the only component of recovery. It means MAT is the foundation on which the rest of the Recovery Kernel framework is built. The supplement stack, the natural reward architecture, the identity work, the trauma processing — all of this is possible when the person is alive and not in active withdrawal. MAT creates that condition.

Medication	Mechanism	Clinical considerations and genomic notes
Buprenorphine (Suboxone,	Partial mu-opioid agonist with ceiling effect —	First-line MAT for opioid use disorder. The ceiling effect makes it much safer than full

Subutex, Sublocade)	activates opioid receptors enough to prevent withdrawal and reduce craving without producing full opioid euphoria at typical doses. Long half-life (24–72 hrs) allows once or twice daily dosing.	agonists — respiratory depression is limited. Buprenorphine naloxone combination (Suboxone) discourages injection. ABCB1 and CYP3A4 variants affect plasma levels — inform prescriber if standard doses feel insufficient or overwhelming. Now available via telehealth in most US states. Sublocade (monthly injection) removes daily compliance variable.
Methadone	Full mu-opioid agonist at clinical doses — completely prevents withdrawal and craving. Extremely long half-life (24–36 hrs) provides stable coverage with once-daily dosing.	Most effective MAT for high-tolerance fentanyl dependence where buprenorphine may be insufficient. Requires daily dispensing at a licensed opioid treatment program (OTP) initially — can be a logistical challenge but saves lives. QTc prolongation risk — baseline EKG and monitoring required. CYP3A4 and CYP2D6 heavily involved in metabolism. Drug interactions are numerous and some are dangerous — pharmacist review of all medications essential.
Naltrexone (Vivitrol — extended release injection)	Opioid receptor antagonist — blocks opioid effects completely. Monthly injection removes daily compliance variable. Produces no opioid effect itself.	Effective option for people with strong motivation who have already detoxed — requires 7–10 days of opioid-free status before initiation (can precipitate severe withdrawal if opioids are present). OPRM1 A118G carriers show strongest response. NOT appropriate as first-line during active opioid withdrawal or for fentanyl-dependent patients who cannot achieve the required abstinence period. Most effective when combined with robust psychosocial support.
Extended-release naltrexone + behavioral support	Same as above — most evidence-based when paired with comprehensive counseling and social support	The EXTEND trial and other RCTs show that naltrexone's outcomes approach buprenorphine when combined with intensive behavioral support. The compliance challenge (the required abstinence period) means it is not appropriate for all patients — careful case selection is essential.

3.2 — The Withdrawal Management Protocol

Whether managing withdrawal as a bridge to MAT, as preparation for naltrexone initiation, or as the detox approach for lower-intensity opioid dependence, the following medical supports are established clinical practice:

Medication/	Role	Notes

intervention		
Clonidine (alpha-2 agonist)	HPA axis suppression — reduces the autonomic storm of opioid withdrawal	Clonidine directly addresses the norepinephrine surge that produces the most distressing physical symptoms of withdrawal: sweating, rapid heartbeat, elevated blood pressure, anxiety, agitation, and insomnia. Not an opioid — does not produce euphoria or treat all withdrawal symptoms. Most effective for the autonomic component. Requires BP monitoring — can cause significant hypotension.
Loperamide (Imodium)	GI symptom control — diarrhea and cramping	Standard dose loperamide addresses the GI symptoms of withdrawal effectively. IMPORTANT: High-dose loperamide has been misused for opioid withdrawal management and can produce cardiac arrhythmias and death at excessive doses. Use at standard OTC dosing (2mg per dose, maximum 16mg/day) under medical guidance only.
Ondansetron / Promethazine	Nausea and vomiting control	Severe nausea is one of the most debilitating withdrawal symptoms. Prescription antiemetics are significantly more effective than OTC options. Ask your physician specifically for nausea management support — it is standard withdrawal management.
NSAIDs (ibuprofen, naproxen)	Muscle aches, joint pain, headache	The diffuse musculoskeletal pain of opioid withdrawal responds to NSAIDs. Take with food to protect gastric mucosa. Contraindicated if renal impairment is present — check with physician.
Hydroxyzine (Vistaril)	Anxiety, insomnia, itching	Non-addictive antihistamine with anxiolytic and sedating properties. Useful for the anxiety and sleep disruption of withdrawal without adding addiction risk. Frequently prescribed as part of withdrawal management protocols.
Lucemyra (lofexidine)	FDA-approved non-opioid withdrawal management	Alpha-2 agonist similar to clonidine, specifically FDA-approved for opioid withdrawal symptom management. Less hypotension than clonidine. Limited to withdrawal management — not maintenance treatment. First non-opioid FDA-approved specifically for this indication.

3.3 — The Withdrawal Timeline

Timeline	Biology	Symptoms	What to do
Hours 6–24 (short-acting	Opioid receptors emptying; HPA axis	Anxiety, yawning, runny nose, tearing,	This is the window where MAT initiation

opioids) / 24–36 hrs (methadone)	beginning to rebound; norepinephrine surge starting	restlessness, early muscle aches	(buprenorphine) is most effective if planned. If managing without MAT: clonidine, hydroxyzine, hydration, warmth. Do not attempt to 'push through' alone if alternatives exist.
Hours 24–72 (peak for most opioids)	Maximum withdrawal intensity; HPA axis in full overdrive; endorphin system fully offline; dopamine system in acute deficit	Severe muscle aches and cramping, vomiting and diarrhea, goosebumps, sweating and chills, insomnia, extreme anxiety, restless leg, profound dysphoria	HIGHEST RELAPSE RISK WINDOW. The suffering is extreme and the body is telling the brain that the solution is one dose away. It is not — it is one dose away from overdose with zero tolerance. Medical supervision, company, and MAT consideration are highest priority.
Days 3–10 (post-acute acute)	Physical withdrawal resolving; emotional and psychological symptoms peaking; endorphin and dopamine systems at floor	Profound depression, anhedonia, emotional rawness, insomnia, mild physical symptoms persisting, powerful cravings	Begin supplement stack in full (Section 4). Exercise as soon as physically capable — even walking. Begin nutrition protocol. Sleep management is critical — poor sleep is the strongest predictor of early relapse in opioid recovery.
Weeks 1–4	Endorphin and dopamine systems slowly recovering; HPA axis normalizing; opioid receptors beginning to upregulate	Gradual improvement with significant variability; PAWS episodes likely; emotional volatility; sleep disruption continues; cravings episodic	Full protocol running. Begin natural reward architecture building. Trauma work can begin gently if stabilized. Community connection is critical in this window.
Months 1–6	Significant receptor upregulation; dopamine system rebuilding; endorphin system recovering; opioid-induced hyperalgesia (amplified pain sensitivity) resolving	Progressive improvement; genuine moments of natural pleasure returning; cognitive clarity improving; pain sensitivity normalizing toward baseline	This is where the work becomes sustainable rather than purely survival. The natural reward architecture begins to provide genuine relief. Community roots deepen. Identity work becomes possible.
Months 6–18+	Mu-opioid receptor density substantially recovered; endorphin system producing natural	Natural enjoyment, connection, and comfort accessible without chemicals; PAWS episodes rare;	Full endorphin system recovery in most people by 12–18 months of sustained abstinence or stable MAT. The person who could not

	comfort and social bonding again; dopamine baseline normalized	episodic cravings contextual rather than constant	imagine feeling this way in month 1 has arrived.

3.4 — Post-Acute Withdrawal Syndrome in Opioid Recovery

⚠ PAWS IN OPIOID RECOVERY CAN LAST 18–24 MONTHS. FENTANYL PAWS MAY BE LONGER. THIS IS NOT WEAKNESS. IT IS NEUROBIOLOGY.

PAWS in opioid recovery is characterized by: waves of anxiety and dysphoria, cravings that appear suddenly without obvious triggers, sleep disruption, emotional numbness alternating with emotional flooding, cognitive fog, and pain sensitivity that fluctuates. Episodes can last hours to days and appear weeks or months after the last use.

The OPRM1 genotype affects PAWS severity — A118G carriers may experience more pronounced social pain and loneliness during PAWS episodes because of the endorphin system's role in social bonding. These episodes are not evidence of failure. They are evidence of a brain recalibrating a system that was severely disrupted.

PAWS interrupt protocol: return to Protocol 01 (Shelter) immediately. Contact your MAT provider if on MAT. Contact your support structure. Execute the craving interrupt sequence (Section 5). Increase magnesium and omega-3. Do not be alone with this.

Section 4 — Biological Reconstruction

Opioid recovery requires the reconstruction of two systems: the endogenous opioid system (endorphins, mu-receptor density, social bonding capacity) and the secondary dopamine system damage. These are addressed in parallel. The methylation foundation underlies both.

4.1 — The Methylation Foundation

Same foundation as every guide in this series — but with two opioid-specific additions: (1) MTHFR variants are significantly overrepresented in opioid use disorder; (2) opioid use itself depletes B-vitamins and disrupts methylation cycles, creating a compounding deficit that must be addressed aggressively in early recovery.

Supplement	Dose	Timing	Opioid-specific notes
5-MTHF (L-methylfolate)	400–1000mcg daily	Morning with food	The baseline neurochemical deficit that opioids were silencing may be partially explained by impaired methylation. 5-MTHF directly supports serotonin, dopamine, and norepinephrine synthesis. Often produces noticeable mood improvement within 1–2 weeks — sometimes the first clear signal that the system is responding to recovery support.
Methylcobalamin (B12)	1000–2000mcg daily	Morning, sublingual	Opioids impair myelin integrity over time. B12 is essential for myelin synthesis and nervous system repair. Sublingual absorption. The neurological repair that B12 supports is particularly relevant for people with significant opioid use histories.
P5P (active B6)	25–50mg	Morning	Cofactor for dopamine and serotonin synthesis. Opioids deplete B6 through multiple mechanisms. Without P5P, the precursor stack below cannot convert to active neurotransmitters regardless of supply.

Magnesium glycinate	400–600mg elemental	Split: morning + evening	Magnesium deficiency worsens pain sensitivity (hyperalgesia), anxiety, sleep disruption, and muscle cramping — all of which are prominent in opioid withdrawal and PAWS. Opioid-induced hyperalgesia is a documented phenomenon where chronic opioid use paradoxically increases pain sensitivity; magnesium addresses one component of this.
Zinc	25–30mg daily	Evening with food	Zinc modulates NMDA glutamate receptors and is involved in endorphin system function. Opioid use depletes zinc. Zinc deficiency worsens depression, immune function, and wound healing in recovery. Take with food to prevent nausea.

4.2 — Endorphin System Reconstruction

THE CENTURION — Rebuilding the System That Provides Comfort, Connection, and Pain Tolerance

The endogenous opioid system cannot be supplemented the way dopamine can be supported with precursors. There are no oral endorphin supplements — the peptides do not cross the blood-brain barrier. What can be done is: provide structural support for receptor upregulation, reduce the inflammatory state that suppresses endorphin production, and — most powerfully — use the behaviors and experiences that trigger endorphin release naturally.

Supplement / intervention	Dose / approach	Timing	Role in endorphin system recovery
DL-Phenylalanine (DLPA)	500–1500mg daily	Morning, away from protein	DLPA inhibits enkephalinase — the enzyme that breaks down endorphins and enkephalins. By slowing the breakdown of your own endorphins, DLPA extends their duration of action. This is the closest available supplement approach to endorphin system support. Particularly useful in the first 3 months when the system is at its lowest. Start at 500mg; assess for stimulating effects.
Omega-3 (DHA + EPA)	3–4g daily, EPA-	With food,	Omega-3 fatty acids are structural components of the

	dominant	morning	membranes in which opioid receptors are embedded. DHA supports receptor membrane fluidity and function. EPA has strong anti-inflammatory effects relevant to both opioid-induced neuroinflammation and opioid-induced hyperalgesia. Essential in the first 6 months.
NAC (N-Acetyl Cysteine)	600–1800mg daily in divided doses	Morning and evening with food	Restores glutamate homeostasis in the nucleus accumbens — the circuit most disrupted by addiction. Multiple clinical trials support NAC for craving reduction across opioid use disorder. Also restores glutathione — the primary antioxidant addressing the oxidative stress of chronic opioid use and withdrawal. Dual-mechanism essential supplement.
Lion's Mane mushroom	500–1000mg standardized extract daily	Morning	NGF stimulation supports neuroplasticity and the formation of new neural pathways — critical when the addiction circuits have been deeply entrenched by chronic opioid use. The 'wanting' circuit's rewiring requires NGF-driven plasticity. Lion's Mane provides this alongside BDNF support.
Uridine monophosphate (UMP) + DHA + choline — the Brain Stack	250–500mg UMP + 1g DHA + 250mg CDP-choline	Morning together	The combination of uridine, DHA, and choline synergistically increases synaptic membrane phospholipids and stimulates D1/D2 receptor synthesis. While developed primarily in the context of dopamine system support, opioid receptor density also benefits from improved neuronal membrane health and phospholipid availability.
Rhodiola rosea	300–600mg (3% rosavins/1% salidroside)	Morning	Adaptogen with endorphin-modulating properties — may have mild enkephalinase-inhibiting effects alongside MAOI activity. Anti-fatigue, anti-stress, mild mood support. Particularly useful in months 1–4 when the endorphin and dopamine systems are both depleted and

			the person's energy reserves are low.

4.3 — Pain Management in Opioid Recovery

THE CENTURION — The Condition That May Have Started Everything Requires Its Own Protocol

⚠ IF CHRONIC PAIN IS THE UNDERLYING DRIVER OF YOUR OPIOID USE DISORDER, REMOVING THE OPIOID WITHOUT ADDRESSING THE PAIN IS NOT RECOVERY. IT IS SUFFERING WITHOUT STRATEGY.

This is one of the most underaddressed aspects of opioid recovery. Standard addiction treatment focuses on the substance. It frequently neglects the pain — physical or emotional — that made the substance necessary. The result is a person in recovery who is also in unmanaged pain, and who is being told that their only option is to suffer through it.

The Recovery Kernel's position: chronic pain in opioid recovery requires a pain management protocol that is as rigorously designed as the recovery protocol itself. The following non-opioid pain management approaches should be discussed with a pain management physician or palliative care specialist:

- Non-opioid medications: SNRIs (duloxetine) and tricyclics (amitriptyline) for neuropathic pain; gabapentinoids for nerve pain (with addiction monitoring); NSAIDs for inflammatory pain; topical agents (lidocaine patches, capsaicin, diclofenac gel) for localized pain.
- Physical modalities: physical therapy, occupational therapy, TENS units, ultrasound therapy, heat and cold therapy, massage, acupuncture (strong evidence for several chronic pain types).
- Mind-body approaches: Mindfulness-Based Stress Reduction (MBSR) has strong evidence for chronic pain and explicitly does not require belief — it changes pain processing neurologically. MBSR also directly addresses the anxiety that amplifies pain perception.
- Movement as medicine: the relationship between movement and pain relief is documented and profound. Even gentle movement reduces inflammation, releases endorphins, and reduces central sensitization. Protocol 03 in the main Compendium (exercise) is not optional for chronic pain in recovery.
- Buprenorphine for pain: buprenorphine is itself an effective analgesic, meaning that MAT and pain management can be addressed simultaneously with the same medication. This is an underutilized option worth explicit discussion with an addiction medicine and pain management physician.

4.4 — Sleep in Opioid Recovery

Opioids suppress REM sleep and alter sleep architecture profoundly. The sleep that opioids produced was not restorative — it was a chemically induced

unconsciousness that prevented the brain from performing the maintenance processes that sleep exists for. In recovery, the return of genuine sleep is both a challenge and a milestone.

- Melatonin: 0.5–1mg sublingual, 30–60 min before sleep. Low dose. Do not use high doses — they disrupt rather than support natural circadian rhythm.
- Magnesium glycinate: 300–400mg elemental evening. Addresses both sleep and the NMDA-mediated hyperexcitability that disrupts sleep in early opioid recovery.
- Glycine: 3–5g, 30 min before bed. Reduces core body temperature; NMDA co-agonist that supports the GABA-glutamate balance disrupted by opioid use; strong sleep quality evidence.
- 5-HTP: 50–100mg evening. Serotonin synthesis support that converts to melatonin. Opioids deplete serotonin — restoring it supports both sleep and daytime mood.
- Phosphatidylserine: 100–200mg evening. Reduces evening cortisol — the HPA axis rebound in opioid recovery produces elevated evening cortisol that prevents sleep onset.
- Tart cherry extract: 480mg or 240ml juice, evening. Natural melatonin source with anti-inflammatory properties relevant to opioid-induced inflammation. Supports sleep architecture without suppressing REM.

Also execute Protocol 17 (Sleep Architecture) from the main Compendium in full.

4.5 — Peptide Support Layer

The following peptides have specific mechanistic relevance to opioid recovery. Physician discussion required — these are research compounds.

Peptide	Mechanism in opioid recovery	Timing	Notes
BPC-157	Dopamine and serotonin system modulation; gut-brain axis repair; opioid receptor modulation in animal models; anti-inflammatory; promotes tissue healing for injection-site damage	Day 1 onwards — daily	BPC-157 is particularly relevant in opioid recovery for two reasons: (1) it has demonstrated opioid receptor modulation effects — specifically counteracting dopamine system depletion from opioids in animal models; (2) people who used injectable opioids often have significant injection site tissue damage that BPC-157 supports. Oral or subcutaneous.

Semax	BDNF and NGF upregulation; cognitive enhancement; dopamine modulation; prefrontal cortex support — specifically the prefrontal circuits that opioids suppress	Weeks 2 onwards	The prefrontal cortex — the decision-making, impulse control, and future-planning center — is heavily impaired by chronic opioid use. Semax's BDNF-driven prefrontal support directly addresses this. The cognitive fog, poor decision-making, and impulsivity of early opioid recovery may respond significantly to Semax. Intranasal.
Selank	Anxiolytic via GABA and serotonin modulation; reduces anxiety without dependence; enkephalin modulation has been suggested in research	Weeks 1–8 — as needed; daily if anxiety is primary symptom	Selank's anxiolytic properties are valuable for the HPA axis rebound anxiety of opioid withdrawal. Its non-addictive profile is particularly important in opioid recovery where substituting one dependence for another is a documented risk. Intranasal.
Epitalon	Sleep architecture; pineal pathway; telomere protection; circadian rhythm normalization	Weeks 1–6 — 10-day cycle	Sleep disruption is one of the most persistent and relapse-driving symptoms of opioid recovery — particularly from fentanyl, where sleep disruption may persist for months. Epitalon directly supports the pineal pathway and circadian biology.
TB-500 (Thymosin Beta-4)	Tissue repair; anti-inflammatory; reduces fibrosis at injection sites; promotes healing of venous damage from IV drug use	Months 1–3 for those with injection-related tissue damage	For people who used IV opioids, the physical damage — collapsed veins, injection site infections and scarring, endocarditis risk — is significant and underaddressed in standard recovery. TB-500 supports tissue repair and anti-inflammatory processes relevant to this specific damage profile. Physician guidance required.

Section 5 — The Opioid Craving — Understanding and Interrupting

THE CENTURION — Opioid Cravings Are Among the Most Powerful Neurological Events in Human Experience. They Are Also Temporary.

Opioid cravings peak at approximately 20–30 minutes and then subside. This is neurological fact. It has been measured. The craving that feels like it will last forever and consume everything has a biological end point. Your only task is to get through the next twenty minutes.

Opioid cravings operate through multiple mechanisms — endorphin-deficit craving (the body missing its pain buffer), dopamine-deficit craving (the reward system seeking any signal), and cue-triggered craving (the most dangerous — environmental triggers that activate the learned response with extreme force).

Cue-Triggered Craving — The Most Dangerous Type

⚠ CUE-TRIGGERED CRAVING IN OPIOID RECOVERY IS NOT THE SAME AS WANTING. IT IS A CONDITIONED NEUROLOGICAL REFLEX. SEEING, SMELLING, OR PASSING THROUGH THE LOCATIONS, PEOPLE, OR SITUATIONS ASSOCIATED WITH USE CAN PRODUCE INSTANTANEOUS, OVERWHELMING CRAVING WITH NO CONSCIOUS DECISION.

This is not a failure of willpower. It is a survival circuit that was trained to fire at maximum intensity. The needle, the spoon, the house, the people, the music — any of these can trigger the full craving response in someone who is otherwise doing well. Environmental redesign (Protocol 05, main Compendium) is not optional in opioid recovery. It is a life-or-death intervention.

THE 5-MINUTE OPIOID CRAVING INTERRUPT — EXECUTE IMMEDIATELY

1. NAME THE CRAVING TYPE. Endorphin-deficit (pain, loneliness, physical discomfort)? Dopamine-deficit (flatness, boredom, anhedonia)? Cue-triggered (specific trigger just fired)? The type changes the response.
2. EXECUTE THE 4-7-8 BREATH. Four full cycles. Now. Not after you finish reading. Now. The parasympathetic response interrupts the automatic quality of the craving-to-action sequence.
3. CHANGE YOUR PHYSICAL LOCATION IMMEDIATELY. If you are near a cue — a person, a place, a situation — leave it. Do not negotiate with the cue. Leave first, process later. Physical distance from the cue environment is not optional.
4. MAKE CONTACT. Call your MAT provider, your sponsor, your recovery contact, or the 988 crisis line. The craving circuit loses power in connection. Say what

is happening. You do not need to have a conversation — you need to be in contact with another person.

5. EXECUTE YOUR ENDORPHIN INTERVENTION. Whatever generates the most endorphin release for you fastest: intense exercise, cold water, physical contact with someone safe, laughter, music at high volume. You are activating the same receptor system through a different door.
6. IF ON MAT: Remember that you took your medication. It is in your system. The craving is a psychological event, not evidence that the medication failed. The medication is working. The craving is the addiction's death throes.
7. DOCUMENT. Trigger, type, intensity (0–10), action, outcome. Three weeks of this data will show you your pattern with a clarity no therapist can achieve through conversation alone.

The Highest-Risk Situations for Opioid Relapse — Know Them Before They Arrive

- Return of tolerance: after any period of abstinence — treatment, incarceration, hospitalization — tolerance is gone. Returning to a prior dose is potentially fatal. Know this before it happens, not in the moment.
- Pain events: dental procedures, injuries, surgeries, and chronic pain flares are documented relapse triggers. Have a pain management plan in place before you need it. Discuss your history with every treating physician.
- Grief and loss: deaths of loved ones, relationship endings, job losses — major grief events produce endorphin-deficit states that trigger opioid craving specifically and powerfully. Have a grief plan that does not involve isolation.
- Celebration: counterintuitively, positive life events are relapse triggers. The brain's association between opioids and 'special occasions' can fire at celebrations, milestones, and successes. Know this about yourself.
- Running out of MAT medication: gaps in MAT due to prescription delays, pharmacy issues, or missed appointments are extremely high-risk periods. Have backup plans for continuity of MAT supply. Missing even one day of buprenorphine or methadone can trigger cravings severe enough to override recovery commitment.

Section 6 — Porting the Addiction: Building Your Own Endorphin System

THE CENTURION — Centurian Paper C-28 Applied to Opioid Recovery

The opioid recovery version of the natural addiction architecture has a specific organizing principle that differs from other substances in this series: the primary target is endorphin release, not dopamine.

The Warrior constitution seeking stimulation needs competition and intensity. The Sage constitution seeking calm needs creativity and contemplation. The opioid recovery constitution — which does not map as cleanly to a single COMT phenotype because opioid use disorder spans multiple profiles — needs: warmth, safety, connection, physical comfort, and pain tolerance. These are the endorphin system's native outputs. These are what must be built.

Natural Endorphin Release — The Architecture

Source	Endorphin intensity	How to access it	For opioid recovery specifically
Sustained aerobic exercise	Very High — 'runner's high' is primarily endorphin-mediated	Running, cycling, swimming, rowing — 30+ minutes at moderate-high intensity	The runner's high is the closest natural analog to the opioid state — warmth, pain relief, emotional calm, and a sense of wellbeing that lasts for hours post-exercise. This is the primary endorphin system activation tool. Non-negotiable. Start where you are — even 10 minutes walking — and build. The endorphin release is proportional to intensity and duration.
Physical human contact	High — oxytocin + endorphin co-release	Safe touch: hugs, handshakes, massage, physical presence with trusted people	Opioids activate the social bonding circuits that human physical contact naturally activates. In recovery, rebuilding the capacity for physical connection — which opioid use disorder frequently destroys through isolation, broken trust, and shame — is not a luxury. It is endorphin system activation through the mechanism the system was designed for.
Laughter	Moderate-	Comedy, absurdity,	Genuine laughter produces

	High — genuine laughter releases endorphins	playfulness — scheduled, not incidental	documented endorphin release. The opioid user who spent years in the anhedonic flatness of tolerance and withdrawal has often lost access to genuine laughter. Rebuilding the capacity to find things funny is neurological repair, not entertainment.
Music	Moderate — chills from music involve endorphin and dopamine release	Music that produces chills or strong emotional response — the specific response is individual	The music protocol in the main Compendium (Protocol 12) addresses this in detail. For opioid withdrawal specifically: 60–75 BPM, warm frequencies, ambient and acoustic — the sonic equivalent of comfort and warmth. The music choices evolve as recovery progresses.
Service and helping others	Moderate-High — sustained; involves endorphin and oxytocin	Sponsorship, peer recovery support, volunteer work, caring for another person or animal	The endorphin release from genuinely helping another person is documented and sustained. For opioid recovery populations specifically — who often come from contexts of profound powerlessness — being useful, being needed, and being capable of alleviating someone else's suffering is among the most transformative available experiences. Many long-term opioid recovery successes cite service as the central pillar.
Cold exposure	High — norepinephrine + endorphin surge followed by sustained calm	Cold shower (last 60–90 seconds cold), cold plunge (2–3 minutes below 60°F)	Cold exposure produces a controlled stress response followed by endorphin release that lasts for hours. For someone whose HPA axis was suppressed by opioids and is now in rebound overdrive, learning to tolerate and then seek controlled stress is neurological skill-building. Start with 15 seconds and build.
Physical accomplishment and mastery	Moderate-High — achievement-related endorphin + dopamine	Strength training milestones, completing physical challenges, learning physical skills	The body that was primarily a vehicle for drug delivery needs to become a source of pride and accomplishment. Physical mastery — getting stronger, learning a martial art, completing a hike, building something with your hands — produces endorphin and dopamine together in the

			context of bodily agency. This is the opposite of what opioid addiction produced.

The Social Reconnection Architecture

THE CENTURION — The Specific Recovery Target That Opioid Use Disorder Attacks Most Brutally

Opioid use disorder destroys social connection with particular ferocity. The drug produces social warmth neurochemically while simultaneously driving behaviors that destroy actual relationships. The result is a person in recovery who is neurochemically primed for connection (because the endorphin system craves it) and socially devastated (because the addiction destroyed it).

Rebuilding social connection is not a therapeutic nicety in opioid recovery. It is an endorphin system intervention. The process:

- Start with recovery community. Twelve-step programs, SMART Recovery, peer recovery support groups — these provide immediate access to people who understand the experience without requiring you to explain or justify it. The social bond formed in shared recovery is unique and powerful.
- Repair the repairable. Protocol 16 (family and relationship repair) in the main Compendium. Not all relationships can be repaired, and not all of them should be attempted in early recovery. But the ones that can be — often are worth the work.
- Build slowly and honestly. The social trust that opioid addiction destroyed was not lost all at once. It will not return all at once. Consistent small actions over months and years rebuild what the addiction destroyed. This is the long game.
- Physical community. Body-based communities — exercise groups, martial arts, hiking clubs, team sports — provide the combination of physical endorphin release and social connection that is the most potent available natural opioid substitute. Both components together are synergistic.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	Opioid-specific notes
Immediate Safety (Before anything else)	Section 0 of this guide; 911; 988	Naloxone access; overdose recognition; fentanyl test strips; Never Use Alone hotline	This phase precedes the Recovery Kernel framework. The Compendium is of no use to someone who does not survive. Harm reduction is first.
Medical Stabilization (Days 0–14)	MAT provider; medical detox if needed; Protocol 01	MAT initiation or detox management; acute withdrawal survival; safety	MAT is the bridge. The Recovery Kernel framework runs alongside MAT, not instead of it. Begin supplement stack as soon as tolerated. Protocol 01 (Shelter) for the acute crisis state.
Biological Foundation (Days 1–21)	Protocol 07, Protocol 09, Protocol 17, Protocol 18	Nutrition, magnesium, thiamine (if alcohol co-use), sleep, gut repair	DLPA on Day 1 if eating. Magnesium on Day 1. Omega-3 on Day 1. The endorphin system reconstruction begins with the structural raw materials it needs. Begin exercise as soon as physically possible.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), Protocol 11	OPRM1 assessment for naltrexone guidance, CYP2D6 for medication safety, MTHFR, COMT	OPRM1 and CYP2D6 are the two highest-priority genetic tests in opioid recovery — they directly affect both treatment selection and medication safety. Get these before MAT optimization if at all possible.
Pain Management (Weeks 1 onwards if pain is a driver)	Protocol 14 (Pain Management), pain specialist consult	Non-opioid pain management; MBSR; physical therapy; buprenorphine for pain	If pain was the entry point to opioid dependence, it requires its own treatment track running parallel to the recovery track. This is not optional. Unmanaged pain is the most predictable path back to opioid use.
Environmental and Social Architecture (Weeks 2–12)	Protocol 02, Protocol 05, Protocol 08, Protocol 13, Protocol 16	Cue removal, addiction transfer prevention, social architecture, relationship repair	Environmental redesign is HIGHEST PRIORITY in opioid recovery — cue-triggered craving is the primary relapse mechanism. Protocol 05 (environmental redesign) must be executed thoroughly and without compromise. People, places, and things associated with use must be addressed.
Trauma Processing	Trauma-specific	Addressing the underlying trauma	Trauma-specific work should not begin until the person is biologically

(Months 1–6 when stabilized)	protocols in Volume IV and V; external trauma therapy	that opioids were managing; EMDR or somatic approaches	stable, on a working MAT regimen if appropriate, and has a support structure in place. Beginning trauma work too early in opioid recovery can destabilize and precipitate relapse. Timing matters.
Identity and Meaning (Months 2–12)	Protocol 20, Protocol 21, C-28	Identity beyond addiction; the person underneath the opioid use disorder; purpose	Opioid use disorder often begins in conditions of profound adversity — trauma, chronic pain, poverty, ACEs. The identity that developed alongside the addiction is not the person. Protocol 20 and C-28 address the reconstruction of identity separate from the using history.
The Long Game (Months 6–24+)	Protocol 24, Protocol 25, C-28, C-31	Purpose architecture; social connection fully rebuilt; endorphin system fully operational	The person in full opioid recovery at 18–24 months, with a functioning MAT regimen or sustained abstinence, a natural endorphin architecture built around exercise and community, and genuine purpose — this person is categorically different from the person who arrived in month 1. This transformation is real. It happens. It is worth the difficulty of getting there.

Section 8 — The Medical Support Layer

Opioid use disorder has the most evidence-supported pharmacological treatment of any substance use disorder. The treatment gap — the percentage of people who need and could benefit from MAT but do not receive it — is approximately 80%. This gap kills people. The information below is intended to help close it.

Co-Occurring Conditions Requiring Direct Treatment

- Chronic pain: requires its own treatment plan. Buprenorphine is an analgesic and may address both simultaneously. Refer to Section 4.3 and Protocol 14 in the main Compendium.
- Depression: extremely common in opioid use disorder, both as a driver and a consequence. MTHFR-variant depression responds to 5-MTHF. Conventional depression may require SSRIs or SNRIs — CYP2D6 status affects metabolism of most antidepressants. Treat the depression as a separate medical condition, not as a recovery phase that will resolve on its own.
- PTSD and trauma: documented in majority of people with OUD. Trauma that is untreated is the most reliable predictor of relapse. Specific trauma treatments (EMDR, CPT, Prolonged Exposure, somatic therapies) have evidence for PTSD in OUD. Timing of trauma treatment matters — stabilization first.
- ADHD: significantly overrepresented in OUD — stimulant use as self-medication for undiagnosed ADHD is a documented pathway. DAT1 and DRD4 variants are relevant. In recovery, non-stimulant ADHD treatments (Strattera, Wellbutrin, Intuniv) are preferred. Stimulant treatment in OUD recovery requires very careful clinical judgment.
- Hepatitis C: blood-borne infection prevalent in IV drug users. Curative treatment now exists (direct-acting antivirals — 95%+ cure rate, 8–12 weeks). Testing and treatment during recovery is standard of care. Curing hepatitis C during recovery improves outcomes and addresses a significant source of ongoing physical illness that complicates recovery.
- HIV: testing and treatment. Undetectable = untransmittable (U=U). Modern HIV treatment does not preclude recovery.

GLP-1 Agonists in Opioid Recovery

The 2025–2026 data for semaglutide and tirzepatide in opioid use disorder shows preliminary trials demonstrating approximately 40% reduction in overdose risk as an adjunct to buprenorphine or methadone. This is not a replacement for MAT — it is a potential add-on that reduces the reward signal from opioids through dopamine and reward circuit modulation. Discuss with your addiction medicine physician.

A Final Word

THE CENTURION — The Pain Was Real. The System That Sought to Silence It Was Doing Its Job. The Work Now Is to Address What Was Underneath.

This guide was written for the person who used opioids not for pleasure but for survival. For the person whose back would not stop hurting, whose nervous system had been wired for terror by things that happened long before the first pill, whose loneliness was a physical ache that the drug was uniquely, powerfully positioned to relieve.

The compendium's dedication is for the ones who ran out of time before anyone told them that what drove them to the substance was biology, not weakness. The opioid crisis has produced more of these losses than any other addiction in modern history. More than 500,000 Americans since 1999. Each one was a person who was managing something real with a tool that was destroying them.

You are reading this because you have not yet run out of time. That is the only fact that matters right now. The biology described in this guide is real and it is recoverable. The endorphin system that went offline comes back online. The mu-opioid receptors that downregulated upregulate again. The dopamine system rebuilds. The pain — physical and existential — can be addressed through means that do not require a substance that will kill you.

Carry your naloxone. Take your MAT. Call someone when the craving comes. Build the endorphin architecture one day at a time. Get through the next twenty minutes.

The Recovery Kernel was built for exactly this journey. Use it.

— — —

NALOXONE: Available without prescription at most US pharmacies. Get it. Give it to people you love.
Never Use Alone Hotline: 1-800-484-3731 | Crisis: 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.
⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE
How to Recover from Opioids Using the Recovery Kernel | Substance-Specific Navigation Series